Specialty Conference

Participants

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Refer to: Lindsay KL, Reynolds TB, Hoefs JC, et al: Ascites—University of Southern California and Rancho Los Amigos Hospital, Downey, California (Specialty Conference). West J Med 134:414-423, May 1981

Ascites

WHEN *this specialty conference was given at the Los Angeles County-University of Southern California* (LAC-USC) *Medical Center Internal Medicine Grand Rounds, the case was presented in three parts using a protocol with stapled pages allowing members of the audience to work through the case in a manner simulating the actual patient's care. Although we are unable to reproduce such a format here, we encourage the reader to pause at the end of each section and formulate his or her own thoughts before proceeding to the next part.*

KAREN L. LINDSAY, MD:* A 56-year-old Mexican-American woman was referred to the LAC-USC Liver Unit on February 4, 1980, for evaluation of ascites.

She had been well until approximately February 1979, when pronounced abdominal swelling developed, which has remained since then. This condition was treated with diuretics, salt restriction and several paracenteses, but each time the fluid was removed, the swelling recurred. There was no pain, only a feeling of distention, some fatigue and mild shortness of breath on exertion while her abdomen was very swollen. Her appetite had remained good. She did not think she had lost any flesh but had not kept track of her weight.

She had never had jaundice, blood transfusions or hepatitis and drank alcoholic beverages rarely.

She had been admitted to a nearby hospital several times for paracentesis. Diagnoses made while she was there included the following: ascites of uncertain cause, mild hypertension, organic heart disease, adult-onset diabetes mellitus and mild renal insufficiency. At that time she was taking the following medications: 0.25 mg of digoxin each day, 50 mg of spironolactone twice a day and 80 mg of furosemide twice a day.

A review of bodily systems gave negative results except for the complaints mentioned above. She stated that she had no urinary symptoms, orthopnea, nocturnal dyspnea, chest pain or edema of the ankles. She had undergone a cesarean section in 1957, an appendectomy in 1964, a cholecystectomy in 1970 and an umbilical hernia repair in 1978.

On examination the patient was obese, with a blood pressure of 124/76 mm of mercury, heart rate of 72 beats per minute, temperature 36.7°C (98°F), respirations 20 per minute, height 144.8 cm (4 ft, 11 in), weight 87.5 kg (192.5 lb). Examination of the skin showed no spider angiomata or palmar erythema. The thyroid gland was not palpable. Neck veins were distended 4 cm above the clavicle at 45° of (head) elevation. She was able to lie flat without shortness of breath, however. On examination of the heart, the point of maximal impulse was not palpable, heart tones were distant but normal and a grade 2/6 systolic ejection murmur was heard at the lower left sternal border. The abdomen was substantially distended with shifting dullness. Old midline and right upper quadrant subcostal scars were present. There were no dilated veins, bruits or masses, and the liver and spleen were not palpable or

No reprints available.
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ABBREVIATIONS USED IN TEXT

Asc:S = ascites to serum ratio
CEA = carcinoembryonic antigen
LDH = Lactic dehydrogenase
$(S\text{-}Asc)_A$ = serum-to-ascites albumin concentration gradient
$(S\text{-}Asc)_{OP}$ = serum-to-ascites oncotic pressure gradient

ballotable. Trace pedal edema was present bilaterally. The remainder of the examination gave normal results.

The hemoglobin was 9.8 grams per dl, leukocyte count 6,200 per cu mm and platelet count normal. The serum albumin was 3.7 and globulin 3.6 grams per dl; alkaline phosphatase 1.6 Bessie Lowry units (normal 1 to 3); total bilirubin 2.0 and direct bilirubin 1.2 mg per dl; aspartate aminotransferase (SGOT) 7 and alanine aminotransferase (SGPT) 7 Sigma Frankel units; lactic dehydrogenase (LDH) 113 units per liter; urea nitrogen 57, creatinine 1.5, uric acid 12.4 and glucose 148 mg per dl. Prothrombin was 67 percent. Serum levels of sodium, potassium, chloride, carbon dioxide, calcium and phosphate were normal.

At this point the reader may wish to pause for a few minutes to consider the possible causes of ascites in this patient and how to proceed in further evaluation. Then Dr. Reynolds will discuss the differential diagnosis of ascites and his approach to the evaluation of a patient with ascites.

TELFER B. REYNOLDS, MD:* The most prominent finding in our patient was ascites, a gross abnormality with a limited number of causes; thus, the cause should always be determined. In this patient there is no doubt about the presence of ascites (Figure 1). With less distention or with an obese abdomen there may be difficulty in deciding whether or not this condition is present. Helpful clues are shifting flank dullness and the patient's recognition that there has been weight gain associated with increased abdominal girth. A large ovarian cyst can simulate ascites but there will be tympany in the flanks due to displaced bowel and dullness centrally. Search for a "fluid wave" is of little value because this physical finding is only present when ascites is tense and obvious. Examination of sodium balance is very useful in assessment of ascites. When ascites is forming, the concentration of sodium in urine is invariably low. There will be a rapid weight gain with ingestion or intravenous administration of sodium, and there may be rapid weight loss with a combination of sodium restriction and diuretic therapy. Diagnostic paracentesis is relatively safe and easy and can be used to prove the presence of ascites as well as to aid in determining its cause. For this procedure, I prefer the left lower quadrant or the midline below the umbilicus for insertion of a 21-gauge needle. One should attempt to avoid injury to the liver, an enlarged spleen, epigastric arteries and veins, visible collateral veins and invisible collateral veins that may be present in adhesions under abdominal scars. Ultrasound testing and computed tomography are noninvasive techniques that are reliable in demonstrating ascites, but it should rarely be necessary to use such expensive tests to settle a relatively simple point.

Although there are many different causes for

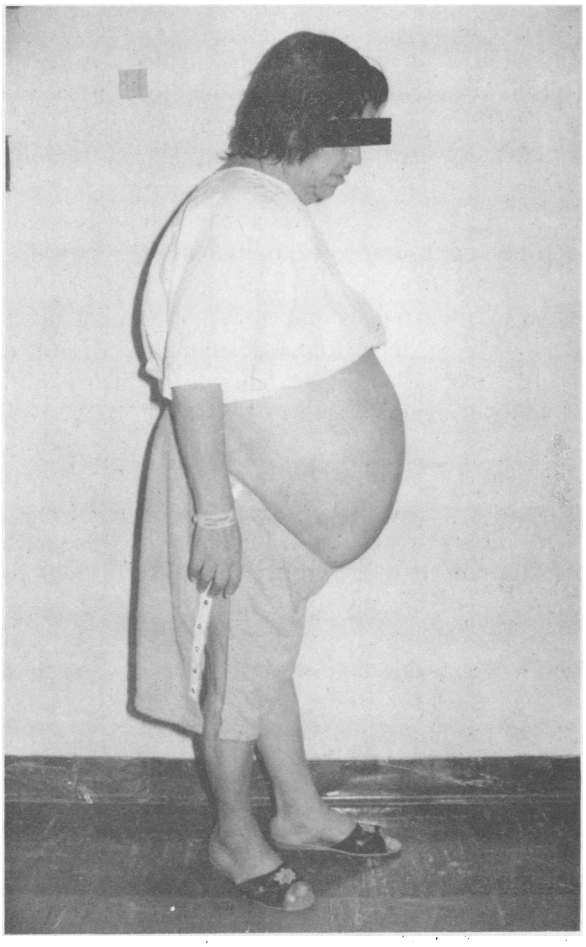

Figure 1.—Side view of patient showing massive ascites and distention of the abdomen.

*Loosli Professor of Medicine.

ascites, our diagnostic acumen may be dulled by the fact that chronic liver disease is by far the most common of these. Therefore, unless we consider *all* the possible causes (Table 1) and routinely carry out diagnostic paracentesis each time we encounter ascites, we are likely to overlook the unusual causes. Some of these are quite treatable.

Peritoneal carcinomatosis as a cause of ascites may be suspected from the clinical history but, more often, is suggested by the findings of diagnostic paracentesis. Protein content is invariably high, the level of lactate dehydrogenase usually is high and the carcinoembryonic antigen (CEA) level may be very high. The fluid may be viscous and feel "slimy." Omental masses are often palpable when the abdominal distention is relieved. Carcinoma can produce ascites by a different mechanism—diffuse embolization of the portal circulation that results in portal hypertension. In such cases the ascitic fluid will resemble that formed in chronic liver disease. This type of ascites may defy diagnosis until evidence of malignancy is found in some other manner.

Cardiac ascites usually is due to constrictive pericarditis or to tricuspid incompetence. Constrictive pericarditis may be particularly difficult to diagnose because ankle edema or significant exertional dyspnea is often absent. The neck veins are the key to diagnosis. Invariably, they are distended in constrictive pericarditis and grossly pulsatile in tricuspid incompetence, if the patient is examined in the sitting position. The protein content of cardiac ascites tends to be high as Dr. Hoefs will explain later.

Pancreatic ascites is caused by leakage from a pancreatic pseudocyst. The only feature that is diagnostic is a high amylase content in the fluid.

Tuberculous ascites is important to recognize because of its treatability. We still see two or three cases annually at LAC-USC Medical Center. Most patients have fever and a high protein content in the ascitic fluid. Culture of the ascitic fluid is sometimes rewarding if a large enough volume is sent for laboratory study. Even more valuable is biopsy and culture of a peritoneal specimen, obtained either by the Cope-needle technique or at peritoneoscopy. Both of these procedures carry some risk because of the frequency of intestinal adhesion to the anterior abdominal wall when there is tuberculous inflammation. In some patients peritoneal tuberculosis is superimposed on chronic alcoholic liver disease; in this situation, the ascites may be caused primarily by portal hypertension in which case the protein content of the fluid may not be high. The diagnosis of tuberculosis is often missed in this type of patient unless a meticulous workup for unexplained fever is carried out.

On rare occasions, *coccidioidomycosis* may cause ascites with similar symptoms and findings as in patients with tuberculosis. The complement fixation test for coccidioidal organisms will be positive and results of peritoneal biopsy may disclose spherules in granulomas.

Occasionally, ascites results from *myxedema.* The clinical diagnosis can be missed only if it is not considered. The protein content of the fluid is invariably high. The relaxation phase of the deep tendon reflexes is grossly slowed.

Nephrogenous ascites is seen in an occasional patient with chronic uremia being treated with either hemodialysis or peritoneal dialysis. There is no satisfactory explanation that I am aware of for nephrogenous ascites and there is nothing diagnostic about the ascitic fluid. Though it is by far the most likely reason for the appearance of ascites in a patient with symptoms of chronic uremia, there may be reason to consider other diagnoses such as chronic hepatitis or tuberculous peritonitis.

Chylous ascites results from rupture of the cisterna chyli or the more distal lymphatic vessels draining the intestine and is usually due to trauma or a malignant lesion. This diagnosis will be made only if the fluid is examined. It will be milky in appearance and the triglyceride content will be several hundred mg per dl or higher.

Pseudochylous ascites is also lactescent but less white in color and contains no excess of triglyceride. Patients with pseudochylous ascites usually have chronic liver disease, and I am unaware of any explanation for the lactescence of the fluid. Differentiation between chylous and pseudochylous fluids often can be made at the bedside by adding petroleum ether to the fluid and shaking it vigorously. With chylous ascites, the triglyceride dissolves in the ether layer and the fluid becomes clear.

Eosinophilic gastroenteritis is a rare cause of ascites, when the process involves the peritoneum. A differential leukocyte count on the ascitic fluid invariably shows a high proportion of eosinophils if the cells are appropriately stained.

Portal vein occlusion sometimes results in ascites, most often after hemorrhage from esophageal

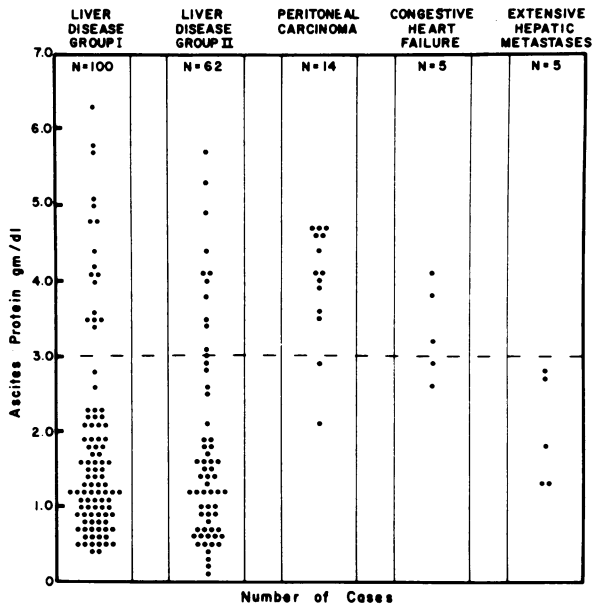

Figure 2.—Protein levels of ascitic fluid. (Reprinted with permission from Boyer T, et al: Archives of Internal Medicine 138:1104, July 1978; copyright 1978, American Medical Association.[2])

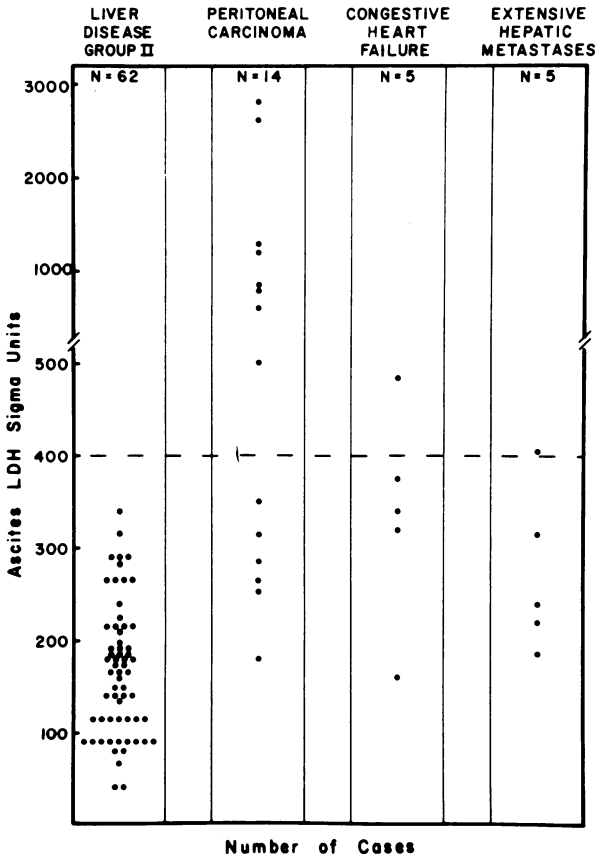

Figure 3.—Lactate dehydrogenase levels of ascitic fluid. (Reprinted with permission from Boyer T, et al: Archives of Internal Medicine 138:1104, July 1978; copyright 1978, American Medical Association.[2])

varices. In such a patient the presence of esophageal varices and the absence of any signs of liver disease may suggest the diagnosis, which can be confirmed with celiac or superior mesenteric angiography.

Hepatic vein thrombosis almost invariably results in ascites. There will be evidence of liver disease, but often a clinician will fail to consider hepatic vein occlusion unless there is right upper quadrant pain, liver biopsy results that indicate intense perivenular congestion or a liver-spleen scan that suggests caudate lobe hypertrophy. In our experience, ascitic fluid protein level is usually similar to that seen in ascites due to chronic liver disease.

Diagnostic paracentesis is the most important tool for determining the cause of ascites and it should be done in every patient in whom there is any doubt about the diagnosis. Light and colleagues[1] found three criteria useful in separating inflammatory from noninflammatory pleural effusions: (1) a pleural fluid-to-serum protein ratio greater than 0.5, (2) a pleural fluid LDH level greater than 200 IU and (3) a pleural fluid-to-serum LDH ratio greater than 0.6. Boyer and coworkers applied the same criteria to the differential diagnosis of ascitic fluid with fairly good results.[2] However, Figure 2, from Boyer's paper, shows the wide range in protein content in ascitic fluid of patients with uncomplicated liver disease and points to the difficulty of using protein content to confirm the diagnosis. Others have noted this same problem previously.[3,4] Later Dr. Hoefs will discuss the control of protein concentration in ascites fluid and will explain how to get maximum diagnostic information from the protein levels.

The LDH level is high in most malignant effusions and is rarely high in other conditions (Figure 3). Ascites leukocyte count is of little value in the differential diagnosis but of great value in pointing to the presence of spontaneous bacterial peritonitis. This is not a *cause* of ascites but, rather, a serious and frequently unsuspected complication of ascites with chronic liver disease. It has also been reported as a complication of ascites due to the nephrotic syndrome in children. A total number of polymorphonuclear cells greater than 250 per cu mm is suggestive enough of spontaneous bacterial peritonitis to justify antibiotic treatment pending the result of cultures. A high total leukocyte count with a low percentage of polymorphonuclear cells is moderately suggestive of tuberculous or carcinomatous ascites; however, the

normal range of leukocytes in ascitic fluid is rather broad in chronic liver disease. Also, as Dr. Hoefs has shown, the total leukocyte count rises with concentration of ascites fluid during diuresis.[5] The carcinoembryonic antigen (CEA) level in ascitic fluid is diagnostic of malignancy if very high (more than 100 ng per ml). The amylase level of ascitic fluid should always be measured because this may be the only clue to pancreatic ascites. Other diagnostic tests that can be ordered on ascitic fluid include cytology, culture for acid-fast bacilli and culture for bacteria. Because they are more expensive, they probably should be requested only when there is clinical suspicion of carcinoma or tuberculosis or when the ascites protein level is elevated.

To summarize the diagnostic approach to ascites—in a woman, always (1) percuss the flanks to look for ovarian cyst; (2) look at the neck veins to rule out cardiac ascites; (3) check the deep tendon reflexes to exclude myxedema, and (4) do a diagnostic paracentesis. After the fluid is obtained, always measure protein content, LD and amylase levels, leukocyte count and differential. Fluid should also be tested for CEA level, bacterial and acid-fast bacilli cultures and cytology if indicated by the clinical findings.

Our patient had ascites for more than a year. A malignant cause seems unlikely because of her relatively benign course. None of the clinical findings or laboratory test results are pathognomonic of chronic liver disease, but this remains a strong possibility. If she has chronic liver disease, it could be cryptogenic cirrhosis or cirrhosis due to hepatitis B. Despite the absence of right upper quadrant pain, she might have the Budd-Chiari syndrome due to hepatic venous occlusion. The duration of her illness is rather long for pancreatic ascites, but this is also a possibility. The presence of neck vein distention at 45° is intriguing and raises the possibility of constrictive pericarditis. Alternatively, she could have both heart and liver disease due to amyloid infiltration or hemochromatosis, or she could have coronary artery disease and cirrhosis coincidentally.

Further evaluation should begin with a diagnostic paracentesis. Serum HBsAg and thyroxine with thyroid-stimulating hormone (TSH) studies would be useful. The neck veins should be inspected closely for Kussmaul sign. A roentgenogram of the chest should be examined for pericardial calcification; cardiac fluoroscopy and echocardiography would also be helpful in assessing the pericardium. A liver-spleen scan would be helpful in deciding whether she has chronic liver disease.

DR. LINDSAY: Roentgenograms of the chest

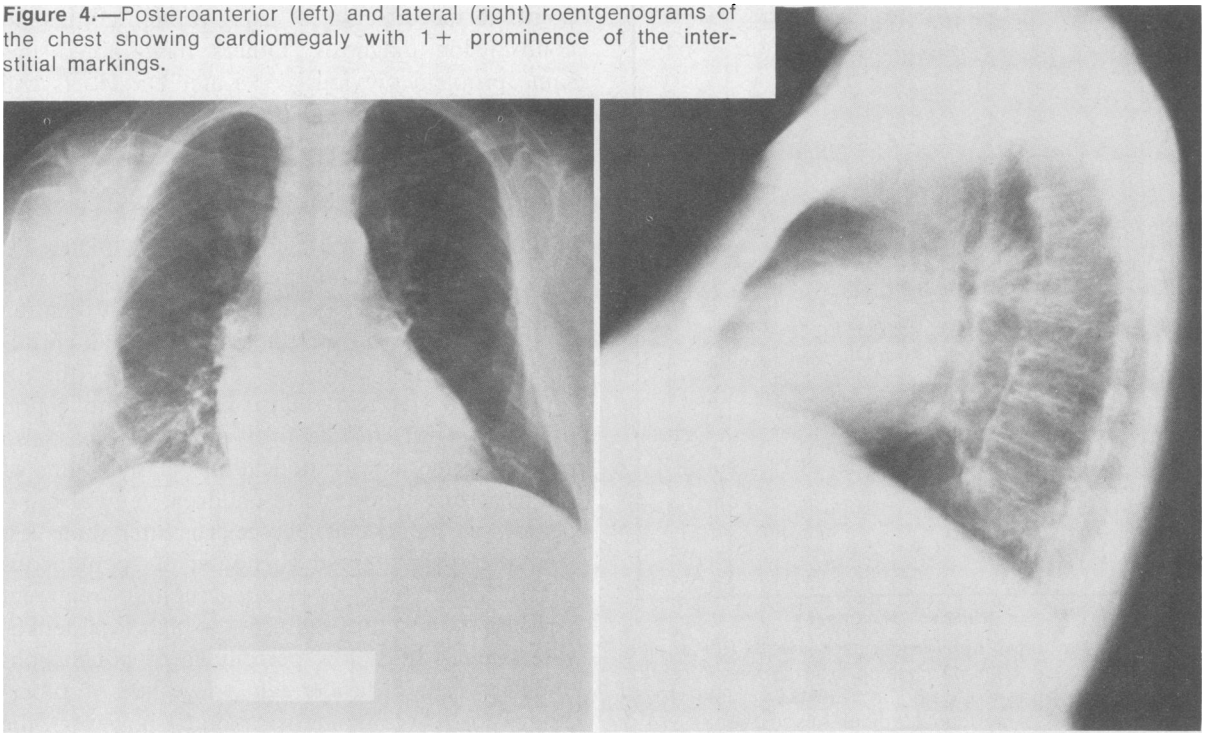

Figure 4.—Posteroanterior (left) and lateral (right) roentgenograms of the chest showing cardiomegaly with 1+ prominence of the interstitial markings.

TABLE 1.—*Causes of Ascites*

Transudative	*Exudative*
Heart failure (tricuspid incompetence or constrictive pericarditis)	Peritoneal carcinomatosis
Inferior vena caval occlusion above hepatic veins	Tuberculous peritonitis
Hepatic vein occlusion	Coccidioidal peritonitis
Veno-occlusive disease of the liver	Pancreatic ascites
Chronic liver disease	Chylous ascites
Acute submassive hepatic necrosis	Myxedema
Widespread metastatic cancer in the liver	Eosinophilic gastroenteritis
Portal vein occlusion	Nephrogenous ascites

showed slight cardiomegaly with 1+ prominence of the interstitial markings (Figure 4). An electrocardiogram showed no abnormalities except for minor ST segment changes attributed to digitalis. Paracentesis disclosed the following: clear straw-colored ascites with 250 leukocytes per cu mm, with a differential count of 91 percent mononuclear cells, 9 percent polymorphonuclear cells and no erythrocytes; albumin 2.7 and globulin 2.5 grams per dl; LDH 87 and amylase less than 80 units per liter.

A technetium 99m sulfur colloid liver-spleen scan showed a diffusely enlarged liver measuring 18 cm in the midclavicular line with slightly diminished colloid uptake in the liver and mild redistribution of colloid activity to the spleen and bone marrow. The spleen was normal in size.

Serum thyroid-stimulating hormone, thyroxine and tri-iodothyronine values were normal. Twenty-four-hour urine sodium level was 11 mEq per liter.

At this point, the reader may wish to pause for a few minutes to consider the potential causes of ascites fluid with these characteristics.

DR. REYNOLDS: The high protein content of the patient's ascitic fluid (5.2 grams per dl) suggested an exudative or inflammatory cause such as tuberculosis or malignancy. Dr. Hoefs will help interpret this finding.

JOHN C. HOEFS, MD:* I will discuss the value of the protein concentration of ascitic fluid in determining the cause of this condition. The causes can be divided into those with vascular outflow obstruction (transudative) and those *without* vascular outflow obstruction and *with* peritoneal membrane involvement (exudative) (Table 1). Several types of ascites do not fit easily into these categories (such as myxedema, chylous ascites and nephrogenous ascites) but are usually considered part of the exudative group. Early studies of ascites suggested that transudative and exudative causes had characteristic protein concentrations.[6,7] This was confirmed in larger surveys,[8-10] which found that the most common transudative process (chronic liver disease) typically had an ascites protein concentration of less than 2.5 grams per dl and most exudative processes (tuberculosis and peritoneal carcinomatosis) had a protein concentration of greater than 2.5 grams per dl. However, these criteria could not separate other transudative sources (cardiac, inferior vena cava block and Budd-Chiari syndrome) from exudative ones. Even ascites secondary to chronic liver disease can have a high protein content; the frequency of high-protein ascites has approached 20 percent in recent studies by Sampliner and Iber,[3] Boyer and colleagues[2] and Bar-Meir and associates.[4] Liver disease (transudative) may be the most common cause of high-protein ascites if these estimates are accurate. The overlap in ascites protein concentration noted in these studies led to the recommendation of 3.0 grams per dl as the dividing line between transudative and exudative processes, but the recent surveys of Boyer and colleagues[2] and Bar-Meir and associates[4] have not found either 2.5 or 3.0 grams per dl reliable diagnostically. The former group[2] recommended multiple criteria similar to those used to separate transudate from exudate in pleural fluid; the usefulness of these criteria awaits further evaluation.

The high protein concentration in ascitic fluid with transudative processes had led to a general disenchantment with the diagnostic value of this determination. This change of attitude was further supported by the recognition that low protein concentrations in ascites resulting from chronic liver disease would increase to above 3.0 grams per dl in most patients during diuresis[5] or albumin infusions.[11-13] Total body extracellular fluid and total body extracellular protein seemed more important determinants of the ascitic protein concentration than the cause of ascites in these studies. It can be concluded that the ascites protein concentration of itself is not an adequate diagnostic tool for separating transudative from exudative causes.

Another diagnostic approach is to consider the relationship between serum and ascites protein

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concentrations rather than the latter concentration alone. Boyer and colleagues[2] found the ascites-to-serum ratio (Asc:S) of protein and LD helpful in diagnosis. However, the (Asc:S) ratio of protein is not specific and increases into the range seen in exudative causes of ascites when patients with liver disease undergo diuresis[5] or albumin infusion.[13] The only relatively stable factor during diuresis, albumin infusion or saline ingestion in chronic liver disease is the serum-to-ascites albumin concentration or oncotic pressure difference.[5,11-14] This gradient is calculated by subtracting the ascites albumin concentration from the serum albumin concentration [$(S\text{-}Asc)_A$] on simultaneous specimens and is thought to be determined by portal vein pressure.[14,15] In a preliminary communication,[16] we reported a good correlation between portal vein pressure and the $(S\text{-}Asc)_A$ in patients with liver disease, confirming this relationship. James[15] and Giges and Kunkel[14] suggested the use of this gradient as an indirect estimate of portal vein pressure. Because exudative causes have a normal portal vein pressure and patients with transudative causes have an elevated portal vein pressure, this gradient from serum-to-ascites albumin concentration [$(S\text{-}Asc)_A$] or oncotic pressure [$(S\text{-}Asc)_{OP}$] contains much diagnostic potential. Giges and Kunkel[14] found a smaller $(S\text{-}Asc)_{OP}$ in patients with exudative sources of ascites than in those with transudative sources, supporting its diagnostic value. In our own experience, the $(S\text{-}Asc)_A$ is equal to or greater than 1.0 gram per dl in 97 percent of patients with chronic liver disease. Patients with ascites of exudative causes (primarily tuberculosis, peritoneal carcinomatosis and pancreatic ascites) have had a value of less than 1.0 gram per dl.

Analysis of data from the few other studies in the literature in which simultaneous ascites and serum albumin concentrations can be abstracted are tabulated in Table 2 and support the potential value of the $(S\text{-}Asc)_A$ in distinguishing ascites of different causes.[5,9,10,13,17,18] As can be seen in Table 2, the predictable difference between portal vein pressure and intra-abdominal pressure for each disease category is related to the serum-ascites albumin concentration gradient. The serum albumin concentration was variable but lowest in cirrhosis and highest in cardiac disease. In cirrhosis, the increase in serum albumin concentration during diuresis was accompanied by a pronounced increase in the protein concentration of the ascitic fluids. A normal serum albumin concentration, a low serum-ascites albumin concentration gradient, or both, were found in all groups with high-protein ascites. Although the serum albumin concentration may be decreased with malignancy, the narrow $(S\text{-}Asc)_A$ results in high-protein ascites. There was little overlap in the

TABLE 2.—*The Serum to Ascites Albumin Concentration Gradient With Cirrhosis, Cardiac Failure and Peritoneal Carcinomatosis**

Author and Reference	*Number of Patients*	*Serum Albumin (grams per dl)*	*Serum to Ascites Albumin Gradient (grams per dl)*	*Expected PVP-IAP Gradient*	*Total Ascitic Protein Concentration (grams per dl)*
Cirrhosis					
Spak[10]	23	2.19	1.83	Increased	1.02
Rovelstad[9]	26	1.86	1.48		1.36
Berendsohn[17]	27	1.89	1.61		1.16
Hoefs[5]	25†	2.36	1.80		1.20
	25‡	3.08	1.43		2.98
Hoefs[13]	10§	2.07	1.44		1.30
	10‖	3.26	1.94		2.44
Cardiac					
Spak[10]	13	3.24	1.56	Normal to increased	3.23
Witte[18] Acute	9	3.67	0.89		5.70
Chronic	3	3.14	1.84		2.80
Peritoneal carcinomatosis					
Spak[10]	86	2.53	0.59	Normal	3.97
Rovelstad[9]	40	2.15	0.45		3.39

PVP-IAP = portal vein pressure—intra-abdominal pressure

*The mean albumin concentrations (serum and ascites) and ascites total protein concentration are abstracted from six studies and listed along with the anticipated portal vein pressure to intra-abdominal pressure gradient.
†Early diuresis.
‡Late diuresis.
§Initial: before albumin infusion.
‖Final: after albumin infusion.

TABLE 3.—*Hemodynamic Findings*

Pressures (mm of mercury)		*Other Determinations*	
Right atrium	22	Cardiac index (L/min/m²)	2.4
Right ventricle ..	50/23	Heart rate	64.0
Pulmonary artery	45/22	Stroke index (ml/m²)	37.0
Pulmonary artery wedge ..	23	End-diastolic volume (ml/m²) .	59.0
Left ventricle ...	130/25	Ejection fraction ..	0.79
Aorta	127/60		

$(S\text{-}Asc)_A$ between transudative and exudative processes regardless of the ascitic protein concentration. We conclude that an $(S\text{-}Asc)_A$ of less than 1.0 gram per dl on simultaneously obtained serum and ascites specimens indicates that portal hypertension is minimal or absent and that ascites results from a condition *other than portal hypertension.*

The $(S\text{-}Asc)_A$ tends to be relatively constant while ascites increases in patients with liver disease because liver blood flow is maintained despite a pronounced increase in intra-abdominal tension with maintenance of a stable portal vein pressure-intra-abdominal pressure gradient.[19]

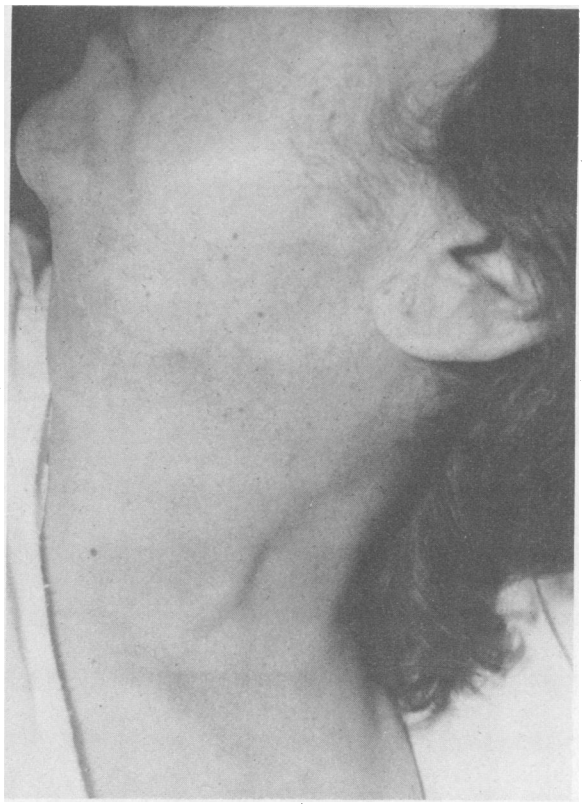

Figure 5.—Neck vein distention at 30° elevation of head of the bed.

However, with cardiac ascites and inferior vena cava block above the hepatic veins, the portal vein pressure remains constant during ascites formation as the intra-abdominal pressure increases. Therefore, with massive or tense ascites the intra-abdominal pressure may increase to the point where the $(S\text{-}Asc)_A$ gradient is relatively small. Cardiac ascites is known to be high in protein concentration. This is explained by the relatively high serum protein concentration and the relatively low pressure difference between the portal vein and ascites when the abdomen fills with fluid and becomes tense.

The serum albumin concentration and ascites protein concentration for the various causes of ascites are listed in Table 2. The $(S\text{-}Asc)_A$ in

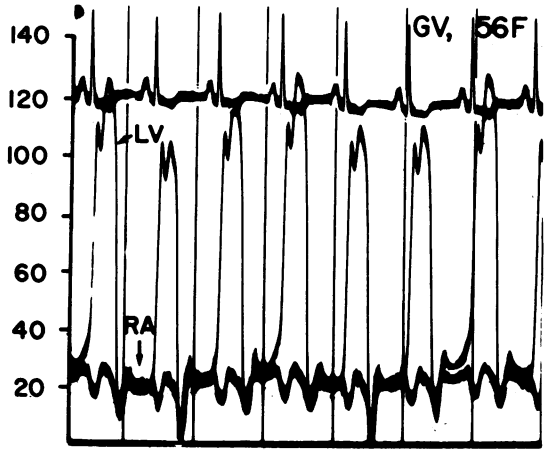

Figure 6.—Left ventricular (LV) and right atrial (RA) pressure tracings. Note the equalization of pressures in diastole.

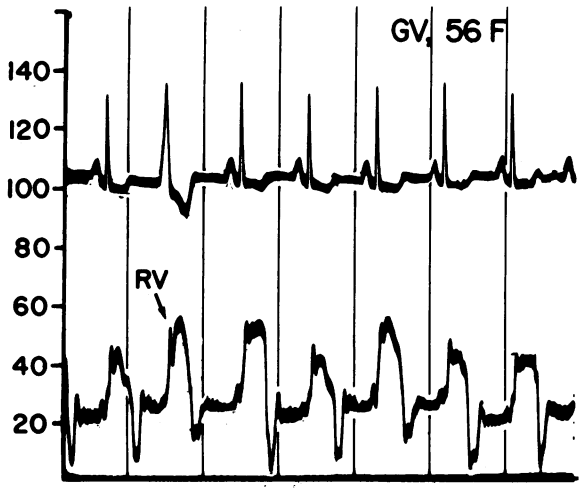

Figure 7.—Right ventricular (RV) pressure tracing showing the "dip-plateau" configuration (square root sign).

our patient pointed away from an exudative process despite the high ascites protein concentration, although the gradient of 1.0 grams per dl did not separate liver disease ascites from cardiac ascites.

We conclude that the ascites protein concentration does not separate transudative from exudative processes. An alternate approach is to assess the presence of portal hypertension indirectly from simultaneous measurement of ascites and serum albumin concentration (or oncotic pressure). Ascites associated with an $(S\text{-}Asc)_A$ gradient of less than 1.0 gram per dl is unlikely to be associated with portal hypertension, thereby eliminating chronic liver disease as the cause. Both exudative and transudative causes of ascites

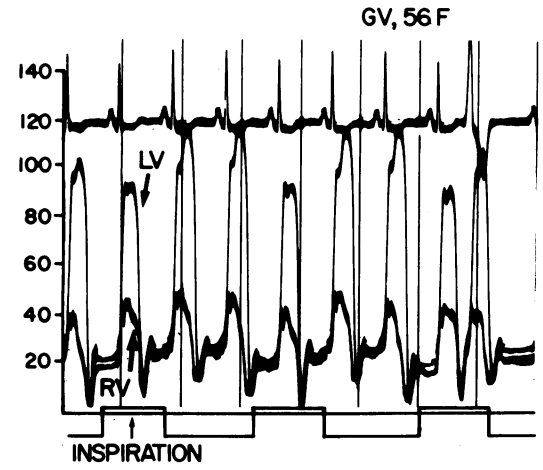

Figure 8.—Left ventricular (LV) and right ventricular (RV) pressure tracings. Note the equalization on diastole, square-root sign and drop in left ventricular systolic pressure during inspiration (paradoxical pulse).

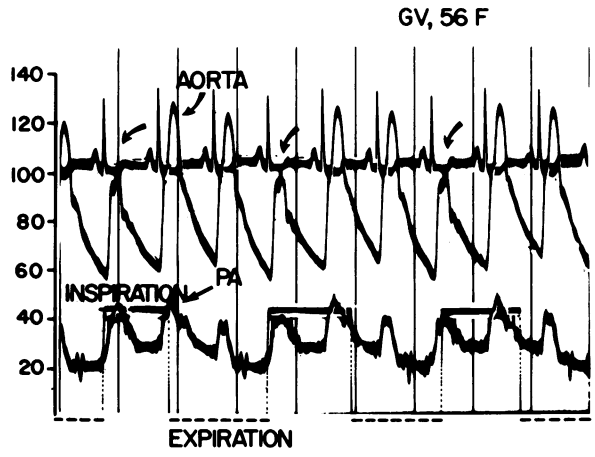

Figure 9.—Ascending aorta and pulmonary artery pressures showing a 20 to 30 mm of mercury paradoxical pulse (arrows).

associated with increased vascular resistance distal to the hepatic veins (cardiac causes and inferior vena cava block above the hepatic veins) could have an $(S\text{-}Asc)_A$ determination of less than 1.0 gram per dl. However, an $(S\text{-}Asc)_A$ of less than 1.0 gram per dl is the rule for uncomplicated exudative causes[9,10] and the exception for cardiac ascites.[10,18] Thus, the $(S\text{-}Asc)_A$ would appear to be of great value in differentiating ascites of unknown causes and should be considered part of the routine evaluation of ascites.

DR. LINDSAY: Because of the consistent finding of neck vein distention on physical examination (Figure 5), constrictive pericarditis was considered, although neither paradoxical pulse nor the Kussmaul sign was detected. Cardiac catheterization was carried out on February 20, 1980.

Dr. Sanmarco will discuss the findings of cardiac catheterization.

MIGUEL E. SANMARCO, MD:* Cardiac catheterization showed findings that were typical of constrictive pericarditis. There was severe elevation and virtual equalization of the left ventricular diastolic, pulmonary arterial wedge, pulmonary arterial diastolic, right ventricular diastolic and right atrial pressures (see Table 3 and Figures 6 and 8).

An early dip-plateau configuration was noted in both the right and left ventricular pressure tracings (Figures 7 and 8), and the left ventricular and aortic pressures showed a 25 mm of mercury drop in inspiration. This paradoxical pulse is clearly seen in Figures 8 and 9.

Left ventricular function was normal as evidenced by normal values for end-diastolic volume and ejection fraction. Selective coronary arteriography detected a left dominant system with only minimal irregularities.

No pericardial calcification was seen; however, there was a substantial gap between the outer portion of the coronary arteries and the edge of the soft tissue density, suggesting increased pericardial thickness or pericardial effusion.

DR. LINDSAY: A tuberculin skin test was done and gave a positive reaction of 11 mm of induration at 48 hours. On March 3, 1980, resection of the pericardium was carried out, at which the pericardium was noted to be 3 to 4 mm in thickness.

Postoperatively, the patient did well. On March

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25, serum urea nitrogen was 28 and creatinine 1.0 mg per dl. On a return visit in August, she reported resolution of exertional dyspnea. Neck vein distention was absent and she weighed 73 kg (160.6 lb).

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Emotional Handling of DES Patients

THE PHYSICIAN, especially the physician who prescribed these drugs, can minimize the regression in the mother most effectively by an approach that deals with the patient as one adult to another. The physician in his own style should state that he appreciates that the experience of knowing of DES exposure is a shock. He should acknowledge what took place by presenting the facts nonjudgmentally. All the physicians who gave this drug should call their patients in and should call the daughters in and say, "Look, we gave you DES and there's a possible problem with the offspring." The feeling should be expressed that the exposure, although worrisome, was given in good faith at a time when medical knowledge indicated that, in order to achieve the pregnancy or in order to hold the pregnancy, this drug was given as an accepted form of medical treatment. Daughters should be approached in the same way. Several of the methods that we do not feel help this whole situation in the daughters are statements such as these: "I'll take care of everything." This infantilizes the woman at a time when she does not need to feel increased dependence. She needs to feel more independent and self-sufficient at this time; "Everything will be OK"—this denies the impact of the news and gives her reassurance that may not be true; it may be premature to say that and it gives her the feeling that she is not being taken seriously and she is being lied to, especially in the face of widespread flood of news on the subject on television and in magazines. "Be quiet, stop crying, take a tranquilizer"—this sort of authoritarian approach will tend to create more anger. . . . And then, "I'll do anything to make it up to you"—and this is generally said by the doctor who is overcome by guilt and blames himself for giving the drug, and as a consequence becomes oversolicitous and overinvolved. Both doctor and patient may lose the sense of what can be done effectively in this sort of a situation.

—LOUIS BURKE, MD, *Boston*

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